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Case Report

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Concomitant pulmonary thromboendarterectomy and supracoronary ascending aorta replacement: a case report

Osman Fehmi Beyazal, Mustafa Karaaslan, Koray Apaydın, Nihan Kayalar, Mehmed Yanartaş

Department of Cardiovascular Surgery, Başakşehir Çam and Sakura City Hospital, İstanbul, Turkiye

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Corresponding Author: Osman Fehmi Beyazal, osmanfehmibeyazal@gmail.com

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ABSTRACT

Pulmonary thromboendarterectomy (PTE) is the treatment of choice for patients with operable chronic thromboembolic pulmonary hypertension (CTEPH). PTE can be performed safely with additional cardiac procedures. However, there are not enough publications in the literature regarding the treatment strategy of CTEPH patients with ascending aortic aneurysms. In this case report, we present a successful case of concomitant PTE and supracoronary ascending aorta replacement.

Keywords: Pulmonary thromboendarterectomy, chronic thromboembolic pulmonary hypertension, ascending aorta replacement

INTRODUCTION

Chronic thromboembolic pulmonary hypertension (CTEPH) is a form of the pulmonary hypertensive disease characterized by incomplete or abnormal resolution of acute pulmonary embolism (PE) causing residual emboli.[1] Pulmonary thromboendarterectomy (PTE) is the treatment of choice in patients with operable CTEPH because of its potential to be curative.[2] PTE can be performed safely with other heart surgeries.[3] However, there are not enough publications in the literature regarding the treatment strategy of CTEPH patients with ascending aortic aneurysms (AAA). In this case report, we present a successful case of concomitant PTE and supracoronary ascending aorta replacement (SCAAR) after the development of CTEPH who was previously treated with thrombolytic therapy for acute pulmonary embolism two years ago.

CASE

A 55-year-old (87 kg) male patient was admitted to our clinic with a complaint of progressive dyspnea. Two years ago, there was a thrombus that started from the proximal part of the right pulmonary artery and extended into the segmental and subsegmental branches of the middle and lower lobes (Figure 1). A catheter was placed in the right pulmonary artery and alteplase (5 mg+0.5 mg/h infusion) was administered. In addition, there was deep vein thrombosis (DVT) in the bilateral popliteal vein and right superficial femoral vein. Transthoracic echocardiography (TTE) showed spontaneous echo contrast (SEC) in the left ventricle and patent foramen ovale (PFO) with marked contrast transition from the right atrium to the left atrium. Systolic pulmonary artery pressure (sPAP) was 55 mmHg, left ventricular ejection fraction (LVEF) was 25%, tricuspid annular plane systolic excursion (TAPSE) was 14 mm, and ascending aorta diameter was 41mm. Afterward, he applied to our clinic again due to the progression of dyspnea in the last 6 months. In TTE, LVEF was 60%, sPAP was 73 mmHg, right heart chambers were dilated, and TAPSE was 19 mm. Pulmonary computed tomographic angiography (CTA) revealed a 16mm thrombus at its widest point extending from the main pulmonary artery to both pulmonary arteries (Figure 1). No critical lesion was detected in coronary angiography (CAG), but a lesion was seen in the left main coronary artery (LMCA) (Figure 2), which was thought to be due to pulmonary bifurcation and right pulmonary artery compression. Intravascular ultrasonography (IVUS) (Figure 2) and coronary CTA (Figure 3) revealed external compression. In addition, the ascending aorta was 47 mm in CTA. As a result of right heart catheterization (RHC); mean pulmonary artery pressure (mPAP) was 78 mmHg, pulmonary vascular resistance (PVR) was 22 W, cardiac output (CO) was 2.8 L/min, and cardiac index (CI) was 1.4 L/min/m^2. On the right lower extremity color doppler ultrasound, no thrombus was observed in deep veins. There were no additional features in his history and laboratory parameters. The patient was evaluated multidisciplinary and it was decided to perform PTE and SCAAR.

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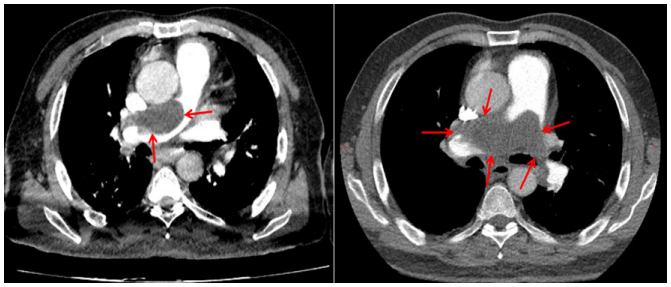

Figure 1. In the left figure, the red arrows indicate the thrombus extending from the proximal right pulmonary artery to the segmental and subsegmental branches of the middle and lower lobes. In the right figure, the red arrows indicate a thrombus extending from the main pulmonary artery to both pulmonary arteries.

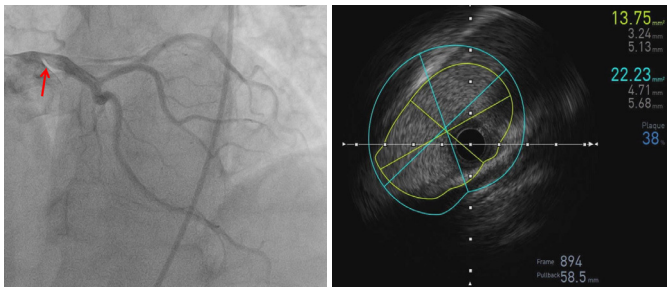

Figure 2. In the left figure, the red arrow shows the lesion at the origin and proximal of the left main coronary artery on coronary angiography. The figure on the right shows an intravascular ultrasonography image consistent with external compression in the left main coronary artery.

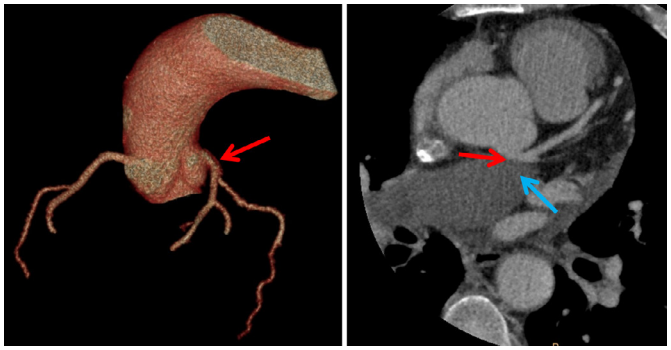

Figure 3. In coronary computed tomographic angiography images, the red arrow in the left figure shows the left main coronary artery. In the right figure, the red arrow indicates external compression by the pulmonary artery to the left main coronary, and the blue arrow indicates the compressing pulmonary artery.

Figure 4. Thrombus removed by endarterectomy from the upper, middle, and lower lobe arteries on the right and from the upper and lower lobe arteries on the left.

After the median sternotomy, aortic and bicaval venous cannulation was performed. Venting cannulas were placed in the pulmonary artery and right superior pulmonary vein. The patient was cooled to 20^{0}C and an arteriotomy was performed on the right pulmonary artery. Level 1 and level 2 lesions were seen at the surgery. After total circulatory arrest (TCA), an endarterectomy was performed on the upper, middle, and lower lobe arteries, respectively, and the arteriotomy was closed (Figure 4). Then, an arteriotomy was performed on the left pulmonary artery. Level 1 and level 2 lesions were seen starting at the pulmonary artery lobe level. Endarterectomy was performed towards the upper and lower lobe arteries at TCA and then the arteriotomy was closed (Figure 4). In the warming phase, the aneurysmatic aorta segment was resected and SCAAR was performed with a 32 mm Intergard graft (Maquet Holding GmbH & Co. KG., Rastatt, Germany). Cross-clamp (XCL) time was 117 min, cardiopulmonary bypass (CPB) time was 335 min, and TCA time was 33 min. He was transferred to the intensive care unit with dobutamine (5 mcg/kg/min). After the operation, there was a total drainage of 575 ml and 1 unit of fresh frozen plasma (FFP) was given. The patient was extubated on the 2nd postoperative day and was discharged on the 7th day with warfarin. As a result of TTE at the postoperative 1st week, LVEF was 40%, sPAP was 20 mmHg, and TAPSE 12 mm. The postoperative PVR could not be seen because the Swan-Ganz catheter could not be placed. He was followed without any complications in the 3 months. Consent form was taken from the patient.

DISCUSSION

It has been reported that PTE may be an option for all patients with CTEPH, including high-risk patients, regardless of the degree of pulmonary hypertension (PH) or right ventricular failure when there is evidence of thromboembolic disease.[4] There are few studies on CTEPH and concomitant cardiac surgeries in the literatüre.[3,5] Although operations such as coronary artery bypass graft (CABG) and heart valve are

frequently reported in these studies, there is no published study on SCAAR to the best of our knowledge. In our case, we present a patient who was given thrombolytic therapy after the development of PE 2 years ago, but subsequently developed CTEPH and additionally underwent SCAAR due to the progression of AAA.

According to the ACC/AHA guideline for aortic diseases published in 2022,[6] there have been some updates in surgical indications compared to previous years. An increase of 3 mm per year in 2 years was determined as the intervention limit, and the intervention limit for the ascending aorta was reduced from 5.5 cm to 5 cm in centers with a multidisciplinary aortic team. In our patient, we decided to replace the ascending aorta because of the 47 mm diameter of the ascending aorta, its enlargement of 6 mm in 2 years, and the need for surgery due to CTEPH.

Additional cardiac interventions during CTEPH surgery increase mortality. It has also been reported that perioperative complication rates are high in patients with high PVR.[3] In our case, the preoperative PVR was 22 W. In addition, the bilateral large amount of thrombus was removed, SCAAR was performed, and the XCL and CPB times were slightly longer than the studies in the literatüre.[3,5] However, there were no complications in the postoperative period, no clinically significant bleeding was observed, and very few blood products were used.

Since our patient is 55 years old, we planned CAG before the operation and we saw a suspicious LMCA lesion. Due to compression of the enlarged pulmonary artery in patients with CTEPH, lesions that appear as stenosis may be seen in LMCA. In the study of Akbal et al.,[7] LMCA compression has been shown to be one of the most important complications of severe pulmonary arterial hypertension. It should be kept in mind that these lesions can be seen in this way due to external compression. IVUS and CTA provide important information in the differential diagnosis.

It has been reported that systemic thrombolytic therapy is the first choice in patients with high-risk PE, and catheter-guided thrombolysis (CDT) may be an alternative in patients who are not suitable for this.[8] However, thrombus may recur over years after thrombolytic therapy, as in our case. Therefore, close follow-up of these patients is extremely important.

In conclusion, patients requiring surgery for CTEPH should be evaluated for other possible cardiac pathologies. For patients requiring additional surgical intervention, this intervention may be appropriately performed during the warm-up period during CTEPH surgery.

CONCLUSION

To the best of our knowledge, our case is the first successful simultaneous PTE and SCAAR operation reported in the literature. These surgeries can be performed concomitantly, safely, and effectively in CTEPH patients with AAA. Cardiac evaluation should be performed in patients with DVT. Close follow-up is important in terms of recurrences after thrombolytic therapy in PE. CTEPH patients may have a stenosis-like appearance in the coronary arteries due to pulmonary artery compression which should be kept in mind in the differential diagnosis.

ETHICAL DECLARATIONS

Informed Consent

The patient signed the free and informed consent form

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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